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Maj Maddry, Joseph

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1100 Willford Hall Loop, Bldg 4430
JBSA-Lackland, TX 78236-9908
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59th Clinical Research Division
1100 Willford Hall Loop, Bldg 4430
JBSA-Lackland, TX 78236-9908
210-292-7141

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Sodium Azide Associated Acute Hyperkalemia in a Swine Model of Sodium Azide Toxicity

PC Ng[1], JK Maddry[1,2], VS Bebarta[3], N Garrett[4], S Boudreau[2], M Castaneda[2], K Canellis[2]

[1]Dept of Emergency Medicine, San Antonio Military Medical Center, San Antonio, TX, [2]CREST Program, Wilford Hall Ambulatory Surgical Center, Lackland AFB, TX [3]University of Colorado-Denver, [4]Geneva Foundation, San Antonio, TX

Background

Sodium azide (NaN3) poisonings are rare but extremely deadly. There is very little in the literature regarding the clinical course of sodium azide poisoning. Virtually all of the information comes from case studies and each of those describe hypokalemia hours after poisoning. Antidotes to cyanide have been used for sodium azide poisonings but have had limited success.

Objective

To describe the clinical course of sodium azide poisoning and develop novel treatments for toxicity.

Methods

Twenty swine (45-55 kg) were anesthetized, intubated, and instrumented with continuous femoral and pulmonary artery pressure monitoring. After stabilization, anesthesia was adjusted such that animals would spontaneous ventilate with an FiO2 of 0.21. Sodium azide, in concentrations ranging from 4 to 160 mg/mL, was infused at doses ranging from 0.8 to 10 mg/kg/min until apnea was confirmed for 1 minute by capnography. This rate was sustained for 1.5 minutes post apnea. Only doses at 10 mg/kg/min at concentrations of 160 mg/mL produced consistent apnea but not sustained apnea.

Methods cont.

Statistics: Repeated measures ANOVA was used to determine statistically significant changes among groups over time.

Results

There were no significant differences in baseline vital signs, chemistries, or arterial blood gases including potassium (mean 4.1 mEq/L) and lactate (1.1 mmol/L) among the animals. Once the NaN3 infusion began, all pigs became hyperkalemic, acidotic and hypotensive. In pigs infused with the highest dose and concentration of NaN3 (n=14), significant hyperkalemia began at apnea (5.1 mmol/L) and continued to rise (mean 7.7 mmol/L) even after the infusion was discontinued. Swine not treated for hyperkalemia died. Those treated with insulin, dextrose 50%, and calcium survived, but demonstrated elevated T waves on electrocardiogram and continued acidosis (lactate mean 6.7 mmo/L).

Table 1. Vital Signs

	Baseline	Apnea	End of Study
pH	7.460	7.403	7.423
pCO2	41.6	50.6	43.1
pO2	96.4	39.4	70.6
K^+	4.1	4.8	6.8
Ca^{2+}	1.28	1.31	1.32
Lactate	1.1	1.5	10.5

Results Continued

Graph 1. Potassium trends over time in treated vs. non treated animals

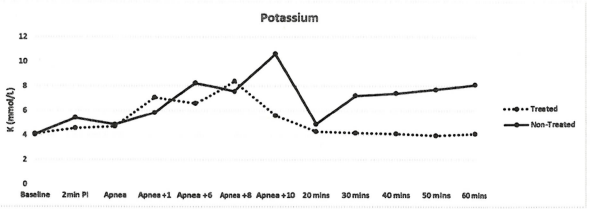
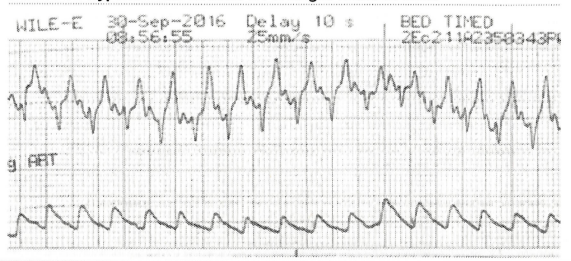


Figure 1. ST elevation in animal #8611. NaN3 started at 0850 and off at 0853. Treated for hyperkalemia at 0852. ST changes noted at 0856.



Limitations

Infusion, not inhalation model
Animal model

Conclusions

NaN3-poisoned swine acutely develop hyperkalemia. We speculate that the hyperkalemia is due, in part, to the intracellular exchange of potassium ions for hydrogen ions in the face of metabolic acidosis. Pathology findings in the animals demonstrate that hyperkalemia is not caused by excessive muscle breakdown. Model development is ongoing.

Corresponding author:
joseph.k.maddry.mil@mail.mil